CLINICAL LECTURES ON CIRCULATORY AFFECTIONS. — LECTURE III. ON ACUTE ENDOCARDITIS.

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(Plate VII.)

You have been taught that the current classification of endocarditis is unsatisfactory from every point of view, and that the most rational subdivision of this affection is a threefold one into the groups—acute, subacute, and chronic. It would be useless to-day to go over the ground which we have traversed so often in the wards as regards the clinical and pathological features of these different types; we shall on this occasion fix our attention entirely upon the acute form of the disease, and my remarks will

be founded upon an instance which you have all had opportunities of observing.

CASE.—M. K., æt. 39, married; was admitted 12th February 1899, to Ward 5A, complaining of pain in the side.

History of present illness.—Two weeks before admission, a sharp pain seized her right side. She had been washing all day in her kitchen, standing over a steaming tub, and going out to the green in a hot perspiring condition, but she felt quite well till the evening. She suffered great pain all night, and stayed in bed but did not call in a medical man till three days after; always thinking the pain would "wear away." She was very breathless, could not lie on her right side, and was very hot and thirsty. Her tongue was extremely dry, and she had a bad taste in her mouth. The doctor told her she was suffering from pleurisy, and applied a mustard leaf to her side. He attended her for a week, applying poultices, and giving her medicine; these relieved her pain greatly. But as she was very weak, and not getting proper nursing, he sent her to the Royal Infirmary.

Past illnesses.—Mrs. K. had always been healthy, and in particular had never had any chest trouble before.

Condition on admission.—The patient lay on her back, looking ill and weak. She breathed somewhat rapidly and with difficulty. Her face was intelligent; she had dark rings round eyes, which were bright and glistening. Respirations, 38–46 per minute. Pulse, 96–108 per minute. The temperature fell almost to normal in the morning, and rose 2° or 3° in the evening.

Alimentary system.—The tongue was large, flabby, and furred, but moist. The patient suffered much from thirst.

Circulatory system.—The heart sounds were regular, but rather weak, with an accentuated sound in the pulmonary region; otherwise they were normal. The pulse was of rather low tension, but was regular in time and force.

Respiratory system.—There was no pain even on coughing, but some dyspnœa. The cough commenced a week ago. It was short, sharp, moist, and not very frequent. The sputum was viscid, mucopurulent; sometimes a slight point of rusty colour was observed. It was found to swarm with diplococci. The right side of the chest did not move much in respiration. Palpation confirmed inspection. The vocal fremitus was slightly increased over the lower part of the right lung and right infraclavicular region, but was diminished over the lateral and anterior parts of the right lung.

A slight feeling of resistance was found on percussion in the right infraclavicular region, and a slightly duller note. Intense dulness was present over the back of the right lung, extending from the base to the inferior angle of the scapula.

In the right infraclavicular region the breathing was bronchovesicular, with medium crepitations on both inspiration and expiration. Over the right supraclavicular region the breathing was harsh vesicular, with prolonged expiration. The vocal resonance was increased. Over the left infra- and supraclavicular regions the breathing was harsh vesicular, with less marked expiration. Over the back of the right lung the breathing was tubular, with medium crepitations on inspiration and

expiration. In some parts at the base the breathing was hardly audible. The vocal resonance was increased, and there was pectoriloquy.

Urinary system.—The urine had a sp. gr. of 1025; it was acid, with excess of urates, while the chlorides were diminished. No albumin was present.

Integumentary system.—The chest was covered with sudamina; there was no herpes.

From the facts of the case, it was obvious that we were dealing with a serious diplococcic infection, resulting in pleuro-pneumonia, with great constitutional disturbance. The temperature rose to a varying height every afternoon during the first few days of her residence in hospital, and began to fluctuate more widely as time went on. The pulse rate varied from 92 to 112, and the respirations numbered between 36 and 44. As the sputum seemed to contain an excessive number of pneumococci, we asked Dr. Welsh to make a culture of it, and he reported that he had seldom seen the pneumococcus so abundant as in our case, and that a rabbit inoculated with the culture died within forty-eight hours, the blood being found to swarm with pneumococci. No other organisms were present.

In spite of stimulant and antiseptic treatment, the patient steadily became worse, and the temperature in the course of a few hours showed variations of many degrees, being as low as 97° in the morning, and almost reaching 106° in the early afternoon. The most sedulous examination of the thoracic organs revealed no change in the condition until the beginning of March, when the exploring needle showed the presence of some pus in the right pleural sac. On aspiration, about 6 oz. of greenish purulent material were withdrawn, and this gave the diplococcus in large numbers. Percussion and auscultation of the heart gave but little change, save some muffling of the heart sounds. During the last few days of her life, it was impossible to examine the condition of the chest thoroughly, on account of her prostration and discomfort. Strychnine, strophanthus, ether, and ammonia were freely used, but the condition steadily became worse, and the patient died on 10th March.

The post-mortem examination was performed by Dr. Welsh, and the following are the abridged notes from his case book:—

The pericardium contained 1½ oz. of turbid fluid, and showed evidence of some recent acute pericarditis. The left pleura was the seat of some scattered fibrous adhesions, but there was no effusion; the right pleural sac contained 20 oz. of purulent fluid.

The heart weighed 13 oz., and exhibited some recent pericarditis. The pulmonary segments presented a few congested points. The aortic orifice was occupied by a large thrombus, measuring about 1½ in. by ¾ in. It was situated at the junction between the right posterior segment and the anterior segment. It was adherent to each of these cusps, as well

as to the endocardium below and the ostium aortæ above. A few scattered patches of atheroma were present at the origin of the aorta. The aortic segments connected with the mass were ulcerated and extensively destroyed. The mitral and tricuspid valves were healthy. The left ventricle was slightly hypertrophied, and the right ventricle somewhat dilated.

The left lung weighed 1 lb., and showed congestion and œdema of the upper lobe, with abundant frothy bronchitis; the lower lobe was hyperæmic and considerably collapsed. The right lung weighed 1 lb. 2 oz. The pleura was extensively adherent, and the whole lung completely collapsed. The pleural covering of the lower two-thirds of the lung was greatly thickened, and showed numerous granulations covered by pus; together with the parietal layer it formed a large empyema cavity. The kidneys were deeply congested, with some advanced cloudy swelling in the cortex.

On microscopic examination of the thrombus, it was found to consist of diplococci with leucocytes and fibrin. No other organisms were present, and but little evidence of organisation could be seen.

Several points of interest arise from the consideration of this case. In the first place, it shows the difficulty of diagnosing certain cases of endocarditis. Although, as you might expect, the possibility of such a complication was considered, no evidence of it was forthcoming, and it was probably a late development in the case. In the next place, it is of interest to observe that the case was an instance of a single infection. No other organism was present in any part of the body. This leads naturally to an expression of regret that we did not make use of the anti-pneumonic serum, which was, at the time when the patient was under observation, coming into use. In any similar case we shall have no hesitation for the future. Many cases of malignant endocarditis are now on record, in which success has attended the use of anti-streptococcic serum, and it is to be hoped that in future anti-pneumonic serum will be found equally satisfactory.

It is unnecessary, in dealing with the case now before us, to discuss the site of the lesion and the apparent reasons for its selection, but it may be well to mention that endocarditis of diplococcic origin is most common in connection with the aortic cusps. It is also needless to pass in review the different secondary lesions which may take place in the course of the affection.

It is highly probable that all cases of acute and subacute endocarditis are of microbic origin, and all may therefore be termed infective. We have still to admit it to be true that cases of endocarditis arising in the course of acute rheumatism have not yet been definitely proved to have their origin in a microbic infection, but it is extremely probable that before long distinct evidence of such a connection will be found. Micro-organisms are found in all forms of endocardial vegetation, and a large number of different microbes have been described in this connection.

Sometimes only one form of organism is present in any given case, but in a considerable proportion of instances of the disease mixed infection has occurred.

Many interesting questions arise as regards the manner in which the micro-organisms attack the endocardium. It must be remembered that the valves differ in respect of their vascularity; while the great venous valves of the heart are freely supplied with blood vessels, the cusps guarding the arterial orifices are destitute of them, unless some previous lesion has led to their development. It therefore follows that endocarditis, when occurring primarily, as in the case which has just been brought before you, on the aortic cusps, must be due to poisons circulating in the blood flowing over the surface of these cusps, and not brought to them by nutrient blood vessels. An important consideration bearing upon this subject is that advanced by Washbourn. The endothelial cells of the vascular system are really phagocytes, capable of englobing bacteria passing along in the circulation. Washbourn quotes the statement of Metchnikoff, that these endothelial cells in fish have their origin in the motile cells on the surface of the yolk sac; hence it is no wonder that they preserve some traces of their ancient activity. That this is true has been clearly proved as regards the blood vessels, and there can be no doubt that the endothelial cells of the heart are endowed with similar functions. In exercising their protective influence by destroying bacteria, these endothelial cells, however, may suffer and give rise to phlebitis or arteritis or endocarditis.

Much work has been done in order to ascertain how organisms can produce endocarditis. Klebs announced his belief in the infection of the endocardium by organisms circulating in the blood and acting directly upon the endothelial surface, while Köster expressed his opinion that the organisms were conveyed by the blood vessels into the endocardium, where they acted by embolic processes. As has just been shown, this view does not account for a primary affection of those arterial valves which are destitute of blood vessels. Wyssokowitsch found that wounding valves by means of a sterilised instrument did not give rise to endocarditis, while the injection of pyogenic organisms into a vein after wounding the cardiac valves gave rise to acute endocarditis. Ribbert went beyond this observation, and showed that the introduction of such pyogenic organisms by means of a vein, without previous injury to the cusps, was a sufficient cause of endocarditis. Since the publication of his results, they have been frequently verified by other observers.

The earliest anatomical change in the development of acute endocardial lesions is the invasion of the endothelial and subendothelial layers by the bacteria of the blood in the cardiac cavities, and this, as has been already remarked, may be regarded as a beautiful example of phagocytic activity. Retrogressive

changes occur in the cells thus invaded, and these lead to the deposition upon the affected surface of fibrin, corpuscles, and platelets. In this way the earliest phase of vegetations is brought about, but proliferation of the cells of the subendothelial layer also takes place, although somewhat later; while leucocytes from the neighbouring blood vessels pass into the affected tissues as well as into the thrombus. As already stated, there is considerable difference in degree between such changes in the venous and arterial valves of the heart. It is probable that such changes occur more or less in all instances of acute and subacute endocarditis, but in the former the cellular infiltration leads to disintegration and abscess or ulceration. In the latter, the newly formed tissues become the seat of a formation of new blood vessels, and their ultimate result is a conversion into granulation tissue. The final result is simple fibrous tissue, which may show fatty or calcareous changes.

A few words may be said on the diagnosis of endocarditis in its more acute form. The recognition of cases of the disease is undeniably difficult. The general appearances are those of an acute infection, sometimes so acute as to bring the life of the patient to a termination on the second or third day of the disease. The temperature is high, but may on the one hand be sustained, on the other fluctuating. The pulse lacks fulness and pressure. It is frequent, often irregular, and sometimes markedly dicrotic. The respiration is hurried and shallow. Copious perspirations are observed. The alimentary system shows appearances similar to those of one of the infectious fevers; the spleen is often enlarged; albuminuria is frequent. Such are the general characters, and according as they are more sustained or more fluctuating, it is common to divide the affection into two types, typhoid and intermittent. In both types, cerebral symptoms are common, and in both of them also embolic processes may give rise to striking features.

When we turn to the physical signs, we find that not infrequently there is a total absence of any characteristic features. The cardiac impulse is usually feeble, and the area of dulness may be increased. In certain cases, changes in the sounds are heard on auscultation. There may be nothing more than a diminution in the intensity of the heart sounds, or they may be muffled in their character, or definite murmurs may be present. Even when murmurs are present, they may not be specially characteristic. As you have been constantly taught, a murmur of regurgitation at the great venous valves does not in itself signify more than a relaxation of the heart wall; when, however, it sets in at an early period of an acute disease, it constitutes presumptive evidence in favour of the probability of endocarditis, for, as Sansom shows, when a mitral systolic murmur is heard at such an early period of any febrile attack as will not allow time for the production of

muscular relaxation, it is probable that the cusps are affected. It must be allowed, however, that in some cases the effects of the poison upon the heart are so severe as to give rise to rapid dilatation.

Since the delivery of this lecture, Poynton and Paine have published the results of their investigations on the bacteriology of acute rheumatism. They have discovered a diplococcus in the blood and tissues, which is found abundantly in the vegetations on the valves. When cultivated, this diplococcus is found to possess the power of setting up acute pyrexia in animals accompanied by endocarditis, and the affected cusps are found to contain swarms of the organism.

Ulcerative endocarditis affecting the aortic cusps